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Inside . . .

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Increased rates of isolation of *Neisseria meningitidis* from blood and cerebrospinal fluid at the ICDDR,B hospital laboratory in Dhaka, 1999-2003.

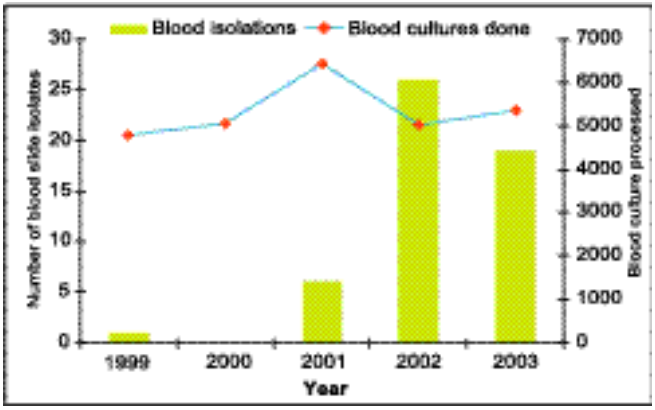
Isolation rates of ***Neisseria meningitidis***, the cause of meningococcal meningitis and meningococcaemia, from blood and cerebrospinal fluid at ICDDR,B have increased dramatically over the five year period, 1999 to 2003. Most cases of invasive meningococcal disease occurred in older children and young adults. Isolates are susceptible to penicillin and ceftriaxone; azithromycin resistance is emerging. Cotrimoxazole was not at all effective. Information from other hospital settings with broader admitting practices and population-based incidence data are needed to more precisely assess trends and burden and to define optimal prevention strategies.

Review of results of culture of blood and cerebrospinal fluid (CSF) at the Clinical Microbiology Laboratory at ICDDR,B indicate that serious infections due to ***Neisseria meningitidis***, serogroup A, are on the rise. Records of blood and CSF cultures processed at ICDDR,B during January 1999 through December 2003 were reviewed. These include cultures from patients hospitalized at ICDDR,B, as well as cultures of specimens referred from other hospitals and clinics in Dhaka. A case of invasive meningococcal disease was defined as a patient from whom ***N. meningitidis*** was isolated from blood or CSF. Serogrouping of strains was done by slide agglutination; isolates were tested for drug susceptibility by disk diffusion (except for cotrimoxazole susceptibility which was assessed by E-test).

During the five-year period, ***N. meningitidis*** was isolated from blood or CSF from 76 patients. Serogroup information was available for isolates from 72 patients; 71

isolates (98.6%) were serogroup A and one was serogroup B. ***N. meningitidis*** was isolated from blood from 53 patients and from CSF from 34 patients—this includes 11 patients from whom ***N. meningitidis*** was isolated from both blood and CSF. Most cases of invasive meningococcal disease (85.5%) were identified during 2002 and 2003, despite similar numbers of tests processed during each of the years 1999 to 2001 (Figure 1). Thus, the rate of isolation from blood cultures in 2002 and 2003 increased nearly five-fold when compared with that in 2001 and >40-fold when compared with 1999 and 2000. Among 1277 CSF cultures processed, 2.5% grew ***N. meningitidis***; likewise the rate of isolation from CSF increased >4-fold in 2002 and 2003 when compared with 2001 and >6-fold when compared with 1999 and 2000. There were no changes in methodology for isolation of bacteria from blood or CSF or in criteria for hospitalization at ICDDR,B during the five-year period of observation.

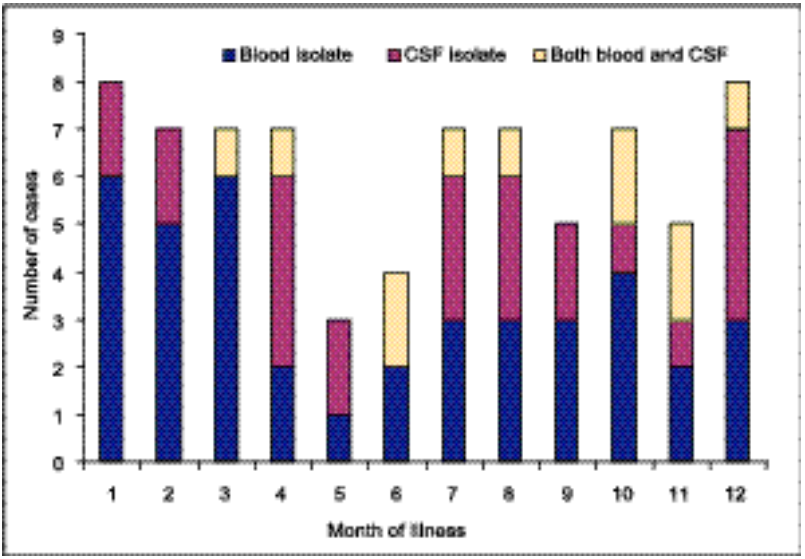
Figure 1: Isolation of Neisseria meningitidis ***from blood cultures, 1999-2003***



Meningococcal disease occurred year round with no obvious seasonal variation, based on the aggregated data (Figure 2). The majority (59.2%) of cases were in patients ≥15 years old and 27.6% of cases were 6-14 years old. All but five patients were younger than 40 years old. Gender information was available for 64 patients; 47 (73.4%) were male.

Gender differences were particularly prominent for patients ≥15 years old among whom 82.5% were male.

Figure 2: Cases of invasive meningococcal diseases by month, 1999-2003



All 83 isolates were susceptible to penicillin, ceftriaxone, and ciprofloxacin. Azithromycin resistance appears to have recently emerged; 27% of isolates from 2003 were resistant, compared with 5% in 2002 and none in 1999-2001. All strains isolated during 2003 were resistant to cotrimoxazole (Table 1).

Table 1: Antimicrobial susceptibility pattern of Neisseria Meningitidis***, 1999-2003***

Year	% Resistant						
	Amp	Chl	PCN	Cip	CTX	Azm	SXT
1999-2001	0	0	0	0	0	0	80
2002	0	0	0	0	0	5	89
2003	0	0	0	0	0	27	100

Amp = Ampicillin; Chl = Chloramphenicol; PCN = Penicillin; Cip = ciprofloxacin; CTX = Ceftriaxone; Azm = Azithromycin; SXT = cotrimoxazole

Reported by: Laboratory Sciences Division and Health Systems and Infectious Diseases Division, ICDDR,B

Supported by: ICDDR,B

Comment

N. meningitidis is a principal cause of bacterial meningitis globally and meningococcaemia is one of the most lethal forms of bacteraemia. Epidemic meningococcal disease can have high attack rates and, especially in sub-Saharan Africa, has had far-reaching, devastating effects (1). Outbreaks associated with large gatherings, like the Hajj, can represent substantial public health challenges because infected people can become ill after returning home, resulting in illness spread to a large number of countries (2). Recent Hajj-associated epidemics have involved ***N. meningitidis***, serogroup W-135, as well as serogroup A (2,3), which was the serogroup responsible for nearly all of the cases identified at the ICDDR,B laboratory.

ICDDR,B hospital admits patients with diarrhoeal disease. This requirement, which has not changed during the time period covered in this report, limits the potential for identifying patients with invasive meningococcal disease; yet, patients with meningococcaemia and meningococcal meningitis were identified in substantially increasing numbers during 2002 and 2003. The findings of this report indicate that ***N. meningitidis*** is becoming an important pathogen in Bangladesh. More information should be collected to determine whether meningococcal disease is occurring with increasing frequency at other hospitals in Dhaka, and elsewhere in Bangladesh.

Effective vaccines are available to prevent meningococcal disease (4)—more information about magnitude of disease burden might suggest a role for vaccine use in Bangladesh.

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Person-to-person transmission of Nipah virus during outbreak in Faridpur District, 2004

From 19 February to 16 April 2004, 36 residents of Faridpur district, Bangladesh became ill with Nipah virus encephalitis; 27 died—this was the second outbreak of Nipah virus encephalitis during 2004. Unlike previous Nipah virus outbreaks, the epidemiologic evidence from this latest outbreak supports person-to-person transmission. At least six patients developed acute respiratory distress syndrome (ARDS), not previously documented as a common feature of Nipah virus infection. Efforts continue to characterize risk factors for infection, enhance infection control measures in hospitals, and understand community beliefs about the virus in order to develop effective prevention messages.

On 5 April 2004 ICDDR,B and the Institute of Epidemiology and Disease Control (IEDCR) were alerted to six cases of lethal encephalitis which had occurred during the previous few weeks among family members and others living within a distinct geographic area in Faridpur district. An investigation began on 6 April at the request of the Ministry of Health and Family Welfare. The team was later joined by scientists from the Centers for Disease Control and Prevention-- Atlanta (CDC), Health Canada, and from Malaysia.

Nipah virus was determined to be the cause of the outbreak through evaluation of sera and other clinical material collected from patients with fever and mental status changes. As a result of surveillance and epidemiologic investigations, ultimately 36 residents of Faridpur District were identified with Nipah-associated illness, including an early case with onset of illness on 19 February (Figure 1); 27 (75%) patients died. Most cases were in adults; four (11%) were ≤15 years old. In addition to fever and mental status abnormalities, (including unconsciousness), cough, respiratory difficulty, headache, and vomiting occurred commonly. Chest radiographs were done in six patients; all showed bilateral infiltrates consistent with acute respiratory distress syndrome (ARDS) (Figure 2).

A laboratory confirmed case was defined by evidence of acute infection demonstrated by the presence of IgM antibodies to Nipah viruses in serum or CSF. A probable case was defined as a patient with fever and mental status changes, who lived or worked in the same village as a confirmed case. Serum specimens were obtained from 27 of 36 cases; 23 were laboratory confirmed. Four of the 27 had undetectable anti-Nipah virus antibody levels. These samples were collected early in the course of illness, perhaps before IgM seroconversion. These patients were categorized as probable cases, as they died before a subsequent sample could be collected for confirmation. An additional nine cases who were linked epidemiologically to laboratory

confirmed Nipah cases died before diagnostic specimens were collected, and they were also designated as probable cases (total number of probable cases=13). Nipah virus RNA was also detected by reverse transcriptase-polymerase chain reaction (RT-PCR) in throat swabs collected from several patients during this investigation.

The preliminary epidemiologic evidence clearly indicates that Nipah virus was primarily spread from person-to-person during this outbreak, perhaps through large droplet transmission. Patients lived in seven different villages, but were clustered in households and families. Prior to their illness onset, 33 of 36 (92%) cases had close contact with at least one person with confirmed or probable Nipah virus infection. Two people (Patients B and C) developed symptoms of Nipah infection seven and 11 days, respectively, after having close contact with their dying brother (Patient A); they also died. Patient A's daughter (Patient D) and her husband (Patient E) became ill six and eight days after a visit to see Patient A late in his illness, and they also died. Patient A became ill on 19 March (Figure 1) and likely transmitted the virus to many other friends and family. Another patient (Patient F) was linked to a variety of subsequent cases. A man who supported Patient F while walking to a different residence during his illness and a rickshaw driver who transported Patient F to a hospital became sick 9 and 10 days, respectively, following one-time exposures; both died. Patient F was initially cared for at his father's house some distance from Patient F's home; patient F's father, and a sister-in law (both of whom cared for patient F) also became severely ill. Patient F and his father died.

Figure 1: Dates of onset of probable and confirmed Nipah cases in the 2004 Faridpur outbreak (N=36)

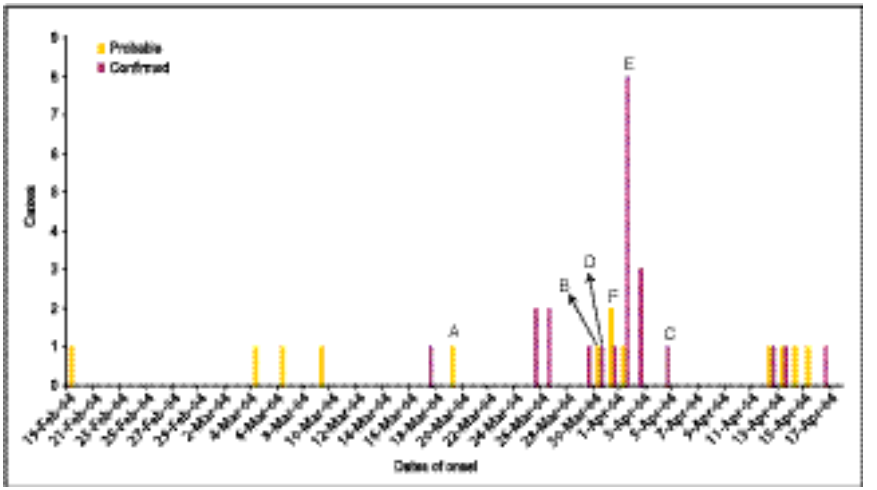
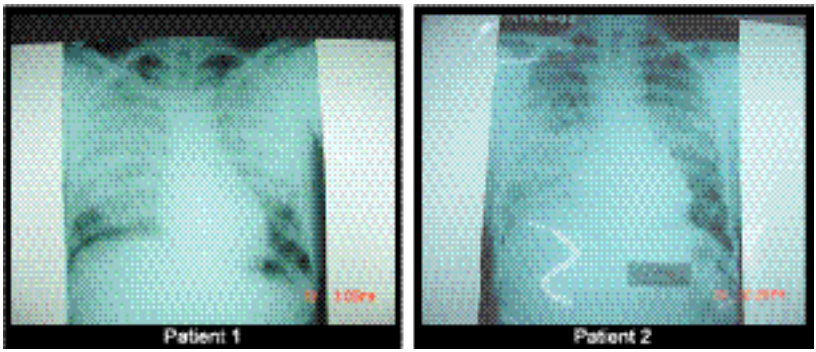


Figure 2: Radiographs from two paients with Nipah encephalitis



The research agenda and investigative strategies developed during this outbreak were based upon the evidence of person-to-person transmission and the need for a long-term approach to prevent and control Nipah virus infection in Bangladesh. Investigation and public health activities included: 1) infection control and patient isolation enhancement; 2) supportive care of patients within local hospitals; 3) a cohort study to determine specific types of behaviours and exposures that may have carried a high risk of transmission of illness; 4) collection and testing of specimens from Indian flying foxes (***Pteropus giganteus***); 5) assessment of environmental surfaces for presence of virus; 6) and behavioural research to characterize perceptions of Nipah-associated illness and healthcare seeking behaviours in the community. The findings of the behavioural research will be used for devising effective health communication strategies and messages.

Reported by: Institute of Epidemiology and Disease Control Research (IEDCR), Faridpur Medical College Hospital, Dhaka Medical College Hospital, Ministry of Health and Family Welfare; Faridpur General Hospital; World Health Organization (WHO); Health Canada; Division of Viral and Rickettsial Diseases, National Center for Infectious Diseases, CDC, USA; Laboratory Sciences Division, Public Health Sciences Division, Clinical Sciences Division, and Health Systems and Infectious Diseases Division, ICDDR,B.

Supported by: Centers for Disease Control and Prevention (CDC), Atlanta, USA; Health Canada, Winnepeg, Canada; Canadian International Development Agency (CIDA), Dhaka; United States Agency for International Development (USAID), Dhaka

Comment

This is the fourth recognized outbreak of Nipah virus encephalitis in Bangladesh, and the second during 2004 (1). The findings of this report suggest that this outbreak was novel in two important ways. First, person-to-person transmission appears to have been the primary mode of spread. Previous outbreaks have been linked to animal sources and person-to-person transmission, while not ruled out, has not previously been clearly documented. (1-3) Second, ARDS occurred in a substantial number of cases during this outbreak, which has not been previously reported as a major symptom of Nipah-associated illness.

Documentation of person-to-person transmission of Nipah virus heightens the need for community containment and rapid isolation/hospitalization of suspect Nipah virus infected patients. Furthermore, appropriate infection control practices, isolation procedures and environmental hygiene within hospitals, are essential. While many cases became infected through caring for family and friends, there were no documented cases of Nipah infection among healthcare workers. There are several possible explanations for the lack of Nipah virus infection among healthcare workers. In the setting of this outbreak, family members, acting as patient attendants, generally had much closer contact with patients than that experienced by healthcare workers. In addition, many patients with Nipah-associated illness did not seek treatment in local hospitals or were hospitalized for very brief periods, limiting the potential for exposure to healthcare workers. Continuing efforts will focus on disseminating infection control protocols to healthcare facilities and developing effective prevention messages for the community, which will address minimizing risk of environmental and human transmission.

Transmission of Nipah virus to humans during this outbreak appears to have been bimodal. Fruit bats continue to be the only identified reservoir for the virus (1-4). During this outbreak, it appears that introduction of the virus into human(s) was followed by person-to-person transmission. Three cases had no known contact with a sick patient before onset of illness and two of these had no known association with other cases. These cases may have been infected through exposure to bat saliva, urine, or faeces, while subsequent cases were infected via exposure to infectious human secretions. During the time of the Faridpur outbreak another active Nipah infection was confirmed in a patient from Rajbari District- the focal point for the previous outbreak in January and February (1). In addition to the two outbreaks documented this year in Bangladesh, isolated, sporadically occurring cases continue to be identified (1).

Nipah-associated illness has emerged as a significant public health problem. While pneumonia, tuberculosis, malaria, and dengue have a much greater public health impact in Bangladesh, the sudden occurrence of several Nipah virus outbreaks with high case fatality rates have had a devastating and frightening impact on families and villages. It appears that there is a zone for epidemic (and perhaps endemic) Nipah virus transmission in western Bangladesh that may be referred to as a "Nipah Belt." Sustained, long term research is needed to characterize the reservoir of the virus and mechanisms for both animal-to-animal and animal-to-human transmission (in particular fruit bat mating and movement patterns, and viral shedding); understand the climatologic and other environmental factors linked to transmission; and to define viral epitopes potentially linked to virulence and transmission. Finally, appropriate infection control practices, hospital hygiene and heightened community awareness about this pathogen and the illness it causes, including how the probability of infection can be minimized, are greatly needed.

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Use of chest and abdominal ultrasound to identify cases of dengue haemorrhagic fever

Surveillance for dengue and dengue haemorrhagic fever (DHF) has shown that ultrasound (US) imaging of the chest and abdomen is a useful adjunct to physical examination and laboratory testing to detect patients with dengue and with criteria of DHF. Patients with criteria for DHF were more likely to have evidence of one or more previous exposures to dengue virus. This is compatible with the theory that exposure to one dengue serotype places individuals at risk for more severe disease upon exposure to other serotypes. Dengue remains a significant public health problem in Bangladesh, requiring implementation of strategies to reduce transmission, and to increase access to health services and supportive care.

Dengue and dengue haemorrhagic fever (DHF) have been recognised public health problems in Bangladesh since 2000 (1). It is likely they were unrecognised problems in earlier years, as well (2). Distinguishing DHF from dengue is important because intensive supportive therapy for DHF can reduce the risk of death and complications. During 2002, ultrasound imaging of the chest and abdomen was added to routine procedures for patients suspected to have dengue at Dhaka Medical College Hospital and Holy Family Red Crescent Hospital (3). The aim was to determine whether this technique is a useful tool for detecting fluid in serous cavities, a criterion for DHF.

Epidemiologic and clinical information was collected systematically from each consenting patient suspected to have dengue. Sera were tested for dengue virus IgG and IgM antibodies by capture ELISA (4). A case of dengue was defined as a febrile patient with ≥40 antibody units by IgG or IgM. An IgM/IgG ratio of ≥1.8 was defined as primary (first time exposure) infection; IgM/IgG ratio <1.8 was defined as secondary infection (previous exposure) (4). DHF was defined as fever with the following: any bleeding manifestations (positive tourniquet test, petechiae, ecchymoses or purpura, bleeding from mucosa, gastrointestinal tract, injection sites, or other locations); thrombocytopenia (100 000 cells per mm^3 or less); and signs of plasma leakage (fluid in chest, abdomen, or pericardial sac) detected by ultrasound in a patient with an antibody titre consistent with dengue infection (5).

Surveillance identified 833 suspected cases of dengue and of these 624 were laboratory confirmed; 3 died (0.48%).

Ultrasound was done on 805 (96.6%) suspected cases of dengue and the following report is based on this group. Of these, 603 (74.9%) patients were confirmed to have dengue antibodies. Most (76%) dengue cases were male.

Consistent with previous findings, dengue cases were more likely to have monthly incomes ≥6000 Taka than non-cases (76.4% vs 68.8%; p=0.03) and also more likely to have >10 years schooling than non-cases (53.9% vs 37.7%; p=<0.001) (Table 1) (3). DHF was subsequently confirmed in 245 (40.6%) cases. Tourniquet test was positive in 490 (81.3%) of dengue cases compared to 93 (47.9%) without dengue (RR=1.7; 95% CI=1.5-2.0).

Accumulation of fluid in serous cavities was detected by ultrasound in 307 (50.9%) dengue cases. Pleural effusion (42.5% vs. 12.4%; p<0.001), ascites (40.3% vs. 18.3%; p<0.001) and thickened gall bladder wall (34.3% vs. 19.3%; p<0.001) were found more frequently among laboratory-confirmed dengue patients compared to patients without laboratory confirmation (Table 1).

Table 1: Ultrasound findings in laboratory confirmed dengue cases compared with patients with similar clinical illness, but without laboratory evidence of dengue

Findings	Dengue (N=603) n (%)	Non-dengue (N=202) n (%)	RR	95% CI	P value
Evidence of plasma leakage[a] by ultrasound	307 (50.9)	44 (21.8)	2.3	1.8-3.1	<0.001
Pleural effusion	256 (42.5)	25 (12.4)	3.4	2.3- 5.0	<0.001
Pericardial effusion	2 (0.33)	0 (0.00)	-	-	-
Ascitis	243 (40.3)	37 (18.3)	2.2	1.6-3.0	<0.001
Hepatomegaly	196 (32.5)	74 (36.6)	0.9	0.7- 1.1	0.282
Splenomegaly	113 (18.7)	33 (16.3)	1.2	0.8- 1.6	0.443
Thickened gall bladder wall	207 (34.3)	39 (19.3)	1.8	1.3-2.4	<0.001

[a]Plasma leakage: any fluid detection by ultrasound either in pleura, peritoneum or pericardium

Primary (first-time dengue virus exposure) infection was suspected (based on antibody pattern) in 157 (26%) cases; secondary infection was suspected in 446 (74%) cases. Patients with secondary infection were more likely to have evidence of pleural effusion (50.7% vs. 19.1%; p<0.001), ascites (46.9% vs. 21.7%; p≤0.001), gall bladder wall thickening (41.7% vs. 13.4%; p<0.001) and hepatomegaly (35.0% vs. 25.5%; p=0.03), when compared with cases with evidence of primary infection (Table 2). Among patients with DHF, 88.2% had evidence of secondary infection compared with 64.2% of patients with dengue (RR=1.3; 95% CI=1.3-1.5; p<0.001).

Table 2: Ultrasound findings among patients with antibody patterns consistent with primary and secondary infection

Findings	Primary N=157 n (%)	Secondary N=446 n (%)	RR	95 % CI	p value
Any plasma leakage	44 (28.0)	263 (59.0)	2.1	1.6-2.7	<0.001
Pleural effusion	30 (19.1)	226 (50.7)	2.7	1.9-3.7	<0.001
Ascites	34 (21.7)	209 (46.9)	2.2	1.6-3.0	<0.001
Thickened gall bladder wall	21 (13.4)	186 (41.7)	3.1	2.1-4.1	<0.001
Hepatomegaly	40 (25.5)	156 (35.0)	1.4	1.0-1.8	0.029
Splenomegaly	29 (18.5)	84 (18.8)	1.0	0.7-1.5	0.920

Platelet counts ≤50,000/mm^3 and a positive tourniquet test were more commonly present in dengue patients with evidence of plasma leakage by ultrasound than in dengue patients without plasma leakage (Table 3).

Table 3: Thrombocytopenia and tourniquet test findings by evidence of plasma leakage among patients with dengue fever

Factors	Plasma leakage (N=307)	No sign of plasma leakage (N=296)	RR	95% CI	p vlaue
Platelate count ≤50000/cmm	178 (58.0)	95 (32.1)	1.8	1.5-2.2	<0.001
Tourniquet test[a] ≥20/sq inch	258 (86.0)	217 (75.9)	1.1	1.0-1.2	0.002

[a]Tourniquet test was refused (stopped in the middle of the procedure due to pain) in 17 cases (N=586, 300 in plasma leakage group, 286 in non plasma leakage group).

Reported by: Dhaka Medical College Hospital, Holy Family Red Crescent Hospital, Laboratory Sciences Division, Health Systems and Infectious Diseases Division and Clinical Sciences Division, ICDDR,B

Supported by: Canadian International Development Agency (CIDA) and Armed Forces Research Institute of Medical Sciences (AFRIMS), Bangkok, Thailand

Comment

While dengue occurred less frequently in 2003 than it had in the three previous years (ICDDR,B unpublished data), it is likely to remain a substantial public health problem for Dhaka and other areas in Bangladesh. Given limited healthcare resources, early indicators for severe disease would be helpful in identifying patients who need more intensive supportive management in

hospital. The findings of this report suggest that detecting fluid in chest or abdomen by ultrasound can identify patients with criteria for DHF.

Previous exposure to ≥1 dengue virus is hypothesized to increase the risk for DHF upon exposure to a dengue virus of a different serotype by a mechanism referred to as antibody-dependent enhancement (6); alternatively, cross-reacting antibodies, possibly linked to over-production of key cytokines, may directly damage capillaries and platelets, resulting in plasma leakage and thrombocytopenia (7). The findings of this evaluation are consistent with these hypotheses—patients with secondary antibody patterns (suggesting previous exposure to dengue viruses) were much more likely to have evidence of plasma leakage by ultrasound than patients without evidence of previous infection with dengue.

Reduction of breeding sources for ***Aedes*** mosquitoes remains the primary method for dengue control (8). Hospitalized cases with dengue tended to have higher socio-economic status than patients with similar febrile illnesses, which were not due to dengue; while more data are needed, this may reflect greater exposure to ***Aedes*** mosquito breeding sources for people with greater economic opportunity. Community education on removing breeding sites should continue to be emphasized in strategies to control dengue in Bangladesh. Experimental vaccines are being developed and may, eventually, provide a way to control this disease.

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Surveillance Update

With each issue of the HSB, updates of surveillance data described in earlier issues will be provided. These updated tables and figures will represent the most recent observation period available at the time of publication. We hope these updates will be helpful to health professionals who are interested in current patterns of disease and drug resistance.

Proportion of diarrhoeal pathogens susceptible to antimicrobial drugs: June 2003-May 2004

Antimicrobial agent	*Shigella* (n=311)	*V. cholerae* O1 (n=630)	*V. cholerae* O139 (n=7)
Nalidixic acid	47.9	NT	NT
Mecillinam	99.0	NT	NT
Ampicillin	47.6	NT	NT
TMP-SMX	35.7	0.2	100.0
Ciprofloxacin	100.0	100.0	100.0
Tetracycline	NT	100.0	100.0
Erythromycin	NT	99.8	100.0
Furazolidine	NT	0.2	100.0

NT=Not Tested

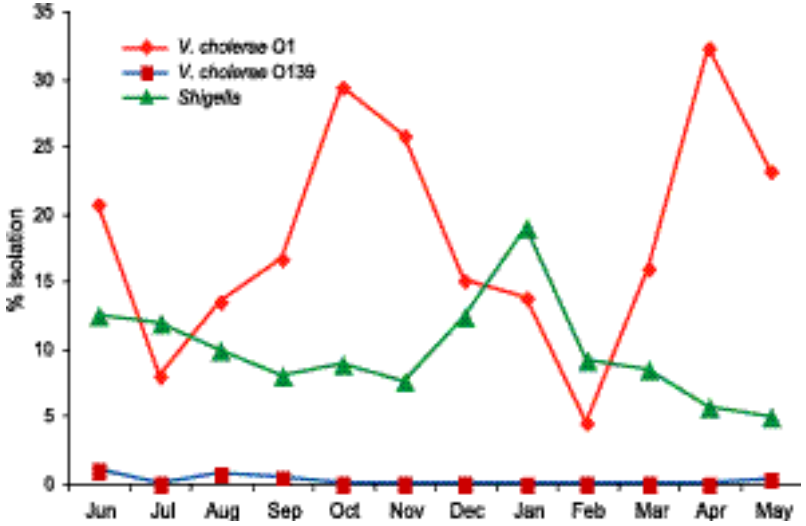
Antimicrobial resistance patterns of 86 M. tuberculosis ***isolates***: ***April-December 2003***

Drugs	Resistance type		Total (n=86)
	Primary (n=69)	Acquired* (n=17)	
Streptomycin	41 (59.4)	10 (58.8)	51 (59.3)
Isoniazid (INH)	10 (14.5)	5 (29.4)	15 (17.4)
Ethambutal	1 (1.4)	2 (11.8)	3 (3.5)
Rifampicin	2 (2.9)	1 (5.9)	3 (3.5)
MDR (INH+Rifampicin)	2 (2.9)	1 (5.9)	3 (3.5)
Any drug	42 (60.9)	10 (58.8)	52 (60.5)

( ) column percentages

* Antituberculous drugs received for 1 month or more

Monthly isolations of V. cholerae ***O1,*** V. cholerae ***O139 and*** Shigella***: June 2003-May 2004***



Antimicrobial susceptibility of N. gonorrhoeae ***isolated during January-March 2004 (n=18)***

Antimicrobial agent	Susceptible (%)	Reduced susceptibility (%)	Resistant (%)
Azithromycin	100.0	0.0	0.0
Ceftriaxone	100.0	0.0	0.0
Ciprofloxacin	0.0	0.0	100.0
Penicillin	0.0	16.7	83.3
Spectinomycin	83.3	16.7	0.0
Tetracycline	0.0	0.0	100.0
Cefixime	100.0	0.0	0.0

ICDDR,B: Centre for Health and Population Research receives financial support from countries and agencies which share its concern for the health problems of developing countries. Current nations providing unrestricted support include: Australia, Bangladesh, Belgium, Canada, Japan, the Netherlands, Sweden, Switzerland, Sri Lanka, the United Kingdom and the United States of America.

Photo : Collecting date-palm juice in Goalanda, courtesy of Ms. Emily Gurley

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